U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 14 October 2005

CASE NO.: 2005-BLA-5376

In the Matter of:

RACHEL WESS, Widow of
JAMES WESS,
Claimant,

v.

CLINCHFIELD COAL CO.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest.

Appearances: Joseph E. Wolfe, Esquire
Andrew Delph, Esquire
For the Claimant

Timothy W. Gresham, Esquire
For the Employer

Before: STEPHEN L. PURCELL
Administrative Law Judge

DECISION AND ORDER DENYING BENEFITS

Statement of the Case

This proceeding arises from a request for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq.* In accordance with the Act and the pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a formal hearing.

A formal hearing was conducted in Big Stone Gap, Virginia on May 9, 2005 at which time all parties were afforded a full opportunity to present evidence and argument as provided in the Act and the regulations issued thereunder, found at Title 20, Code of Federal Regulations.

During the hearing, Director's Exhibits Numbers 1 through 42 (hereinafter referenced as "DX"), and Employer's Exhibits Numbers 1 through 6 (hereinafter referenced as "EX") were received in evidence. Hearing Transcript (hereinafter referenced as "Tr.") 7. At the hearing, Employer withdrew the medical opinion of Dr. Castle as an Employer's Exhibit and substituted the medical opinion of Dr. Naeye as EX 1. (Tr. 10). All of this evidence has been made part of the record. The Claimant did not offer any exhibits aside from those already contained in the Director's exhibits.

At the conclusion of the hearing, the parties were offered an opportunity to submit written closing arguments. (Tr. 22-23). Claimant and Employer filed written briefs on September 27 and 28, 2005, respectively.

Issues

The issue presented in this case is whether pneumoconiosis caused, contributed to, or hastened the death of James Wess, the deceased husband of Rachel Wess.

Procedural History

James Wess, (hereinafter "Miner"), filed an application for benefits on October 19, 1980. (DX 1). After a formal hearing held before Administrative Law Judge V.M. McElroy, that claim was denied by Decision and Order dated June 27, 1988 based on a determination that the Miner had failed to establish total disability due to pneumoconiosis. Judge McElroy's decision was subsequently affirmed by the Benefits Review Board on March 28, 1990, and the Board's decision was thereafter affirmed by the Fourth Circuit on October 31, 1990. *Ibid*.

The Miner died on August 6, 2002 and on November 4, 2002, Rachel Wess, the Miner's widow, (hereinafter "Claimant"), filed an application for survivor's benefits. (DX 3). The Director, Office of Workers' Compensation Programs issued a Proposed Decision and Order awarding the Claimant benefits on May 7, 2004, finding that the Miner had complicated pneumoconiosis. (DX 30). On May 17, 2004, the Employer requested a formal hearing. (DX 32, 33). This matter was thereafter referred to the Office of Administrative Law Judges on December 7, 2004 for a formal hearing. (DX 40).

Findings of Fact

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, testimony presented, and arguments made. Where pertinent, I have made credibility determinations concerning the evidence.

Survivorship and Augmentation

The Miner was born on September 10, 1919, and he died on August 6, 2002. (DX 3, 9). Mrs. Wess was married to the Miner on July 2, 1949, and she remained married to him until his

death. (DX 3, 8). She has not remarried and has no dependents for purposes of augmentation of benefits. (DX 3).

Length of Coal Mine Employment

Employer has stipulated to at least 23 years of coal mine employment by the Miner. (Tr. 5, 6). The Director found that the Miner had 23 years of coal mine employment. (DX 30). The Miner's Social Security Earnings Statement also shows 23 years of coal mine employment. (DX 5). Based upon the documented evidence of record, I find that James G. Wess was a coal miner within the meaning of § 402(d) of the Act and 20 C.F.R. § 725.202 of the regulations for at least 23 years.

Responsible Operator

The evidence reveals that the Miner last worked as a coal miner for a period of at least one year for Clinchfield Coal Co. (DX 5). The Miner's Social Security Earnings Statement, as well as the employment history completed by Claimant, show employment with that company from May 5, 1959 to September 10, 1980. (DX 4, 5, 14). Accordingly, I find that Clinchfield Coal Company was properly designated as the responsible operator in this case.

Applicable Procedural Law

Because this claim was filed after the enactment of the Part 718 regulations, entitlement to benefits will be evaluated under the Part 718 standards. 20 C.F.R. § 718.2. In order to establish entitlement to benefits under Part 718, Claimant must establish by a preponderance of the evidence that the Miner's death was due to pneumoconiosis. *See generally Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994).

Death Due to Pneumoconiosis

Section 718.205 provides that benefits are available to eligible survivors of a miner whose death was due to pneumoconiosis. An eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that the miner's death was due to pneumoconiosis;

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or where death was caused by complications of pneumoconiosis; or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a substantially contributing cause of a miner's death if it hastens the miner's death. 20 C.F.R. § 718.205(c)(5).

In a survivor's claim, a threshold determination as to the existence of pneumoconiosis must first be made. *See* 20 C.F.R. §718.205(a)(1); *Trumbo v. Reading Anthracite Co.*, 17 BLR 1-84 (1993). The medical evidence is set forth below.

MEDICAL EVIDENCE

A. CT Scan Evidence

At Employer's request, Dr. William W. Scott, Jr. analyzed an October 13, 2001 CT scan on April 7, 2005, noting that it consisted of 7 mm sections through the chest displayed at lung and mediastinal settings. (EX 3). Dr. Scott is a board-certified radiologist and qualified as a B-Reader. He found infiltrates and possible fibrosis in both lung bases and minimal infiltrate in the superior segment of the right lobe. Dr. Scott noted that these changes might be pneumonia related to chronic aspiration or could be due to collagen vascular disease. He also found a few calcified granulomata in the lungs, mediastinum and spleen compatible with healed tuberculosis or histoplasmosis. He also noted calcification of the aorta and coronary arteries. Dr. Scott concluded that the CT scan showed no evidence of small, rounded opacities to suggest silicosis or pneumoconiosis. (EX 3, 4).

B. Autopsy Evidence

Dr. Muljibhai J. Turjman performed an autopsy on the Miner on August 8, 2002 (DX 10). Dr. Turjman is board-certified in Anatomic and Clinical Pathology and Cytopathology. The examination included both an internal and external examination. Both lungs were dissected out of the body. Dr. Turjman performed serial sections of the lung tissue and noted:

> There is extensive emphysematous changes involving both lungs, especially both upper lobes with lung tissue exhibiting spongy appearance. Multiple coal macules are present especially in both upper lobes. [A] few, scattered coal micronodules (less than 0.7 cm) are also present. A single white, noncalcified nodule is noted in the left upper lobe measuring 1.2 cm in the greatest dimension. Both lungs are markedly edematous and congested. . . .

(DX 10 at 3). Dr. Turjman also performed a microscopic examination of the lung tissue. He noted:

> The sections of both lungs show diffuse emphysematous changes involving both lobes, but more pronounced in the upper lobes characterized by marked destruction of alveolar spaces forming larger spaces in a picture of panlobular emphysema. Multiple coal macules are present characterized by clusters of pigment-containing hystiocytes present in the vicinity of bronchioles and small bronchi and in the vicinity of small and medium-sized blood vessels. In addition, there are a few scattered coal nodules best manifested in the slides labeled 10, 11

> and 12 of the left upper lobe where a background of collagen is heavily infiltrated by histiocytes containing coal dust with adjacent emphysematous change in the lung tissue. Both lungs, especially the lower lobes, show extensive accumulation of neutrophils within alveolar spaces and within the lumina of the bronchi. The neutrophils are admixed with amorphous fibrinoid material and debris. Areas of early consolidation are noted including the nodular area that is detected grossly in the left upper lobe that microscopically reveals large area of accumulation of neutrophils. No granulomatous inflammation is presented.

Id. at 4. Under the heading "Final Note" Dr. Turjman wrote:

> Examination of the lungs shows moderately advanced features of coal workers' pneumoconiosis manifested by the presence of multiple coal macules and a few scattered coal micro-nodules in association with extensive and diffuse emphysematous changes. The presence of extensive bronchopneumonia in both lungs most likely represents the terminal events in this patient with the underlying coal workers' pneumoconiosis being a contributing factor.

Ibid.

C. Narrative Medical Evidence

Dr. Joshua A. Perper

Dr. Perper, who is board-certified in Anatomical and Surgical Pathology, as well as Forensic Pathology, reviewed a variety of records including the Miner's death certificate, treatment records from 1995 through 2002, fifteen autopsy slides, and the autopsy report prepared by Dr. Turjman. (DX 26). He prepared a report dated December 26, 2003 in which he set forth his findings and conclusions based on that evidence. Dr. Perper's microscopic examination of the autopsy slides revealed multiple birefringent silica crystals and occasional fibro-anthracotic and anthraco-silicotic mirconodules in the pleura, as well as anthracotic macules around blood vessels and airways scattered throughout the pulmonary parenchyma. He also noted micronodules between 0.2 cm and 0.5 cm in size scattered throughout the pulmonary parenchyma, describing them as "primarily of the fibro-anthracotic mixed dust type, although a few resemble the silicotic type." *Id*. at 22-23. Dr. Perper found a single "irregular macronodule exceeding 2.0 cm in maximal dimension and fulfilling the requirement for complicated coal workers' pneumoconiosis." *Id*. at 23. As a result of his examination, he diagnosed:

1. Coal workers' pneumoconiosis with macules, micronodules and macronodule measuring more than 2.0 cm. in maximal dimension, consistent with complicated coal workers' pneumoconiosis (PMF>)

2. Centrilobular emphysema, severe

3. Acute bronchopneumonia, extensive, severe with microabscesses

4. Foci of organizing and organized pneumonia

5. Sclerosis of intra-pulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale

6. No granulomas present

Ibid. He further concluded that: (1) the Miner had objective findings diagnostic of significant coal workers' pneumoconiosis; (2) the fact that the Miner did not smoke for many years prior to his death reinforced the causal relationship between his exposure to mixed coal dust containing silica and his coal workers' pneumoconiosis and emphysema; and (3) complicated coal workers' pneumoconiosis was a substantial contributory cause of the Miner's death, and a hastening factor in his death, both directly and indirectly. *Id*. at 25-29.

Dr. P. Raphael Caffrey

Dr. Caffrey, who is board-certified in Anatomical and Clinical Pathology, reviewed the August 8, 2002 autopsy report of Dr. Turjman, examined fifteen autopsy slides and other medical evidence, and prepared a consultation report dated March 13, 2003. (DX 23). Based on this evidence, he concluded there was no evidence of complicated pneumoconiosis and diagnosed, *inter alia*, acute necrotizing bronchopneumonia bilaterally, moderately severe centrilobular and panlobular emphysema, and simple coal workers' pneumoconiosis (noted as "minimal"). In his report, Dr. Caffrey wrote, in relevant part:

> It is my opinion from a review of the documents I received, the autopsy report and the autopsy slides that Mr. James Wess had a very mild degree of simple coal workers' pneumoconiosis. This very mild degree of simple CWP certainly would not have caused the patient pulmonary disability because the autopsy slides show that the lesions of CWP occupy approximately 2% of the lung tissue.

Id. at 2-3. He further opined that the Miner had moderately severe centrilobular and panlobular emphysema caused by his 40 years of smoking, and that the cause of the Miner's death was extensive necrotizing pneumonia involving all lobes. With respect to Dr. Turjman's diagnosis of moderately advanced coal workers' pneumoconiosis, Dr. Caffrey wrote:

> I definitely disagree with the autopsy pathologist, ... The autopsy slides show minimal or very few lesions of simple CWP, although there are lesions of CWP. My microscopic opinion definitely coincides with what Dr. Turjman's provisional anatomical diagnosis was and that is and I quote: "Possible mild (early state) coal workers' pneumoconiosis."

Id. at 3. (bolding and italics omitted). Dr. Caffrey concluded, based on all the evidence he reviewed, that the Miner's coal mine employment did not cause any pulmonary disability and did not cause, contribute to, or hasten his death. *Ibid*.

According to a supplemental report dated October 7, 2003, Dr. Caffrey reviewed

additional medical evidence including the report of examination prepared by Dr. James Castle on April 7, 1981, Dr. Castle's deposition of April 16, 1985, treatment records of Dr. German Iosif from 1985 through April 2002, medical records from the Clinch Valley Medical Center from 1999 through 2002, and a discharge summary from Wellmont Holston Valley Medical Center dated August 6, 2002. (DX 23). Based on his review of this additional evidence, Dr. Caffrey's opinion remained unchanged.

In another supplemental report dated April 3, 2004, Dr. Caffrey noted that he had reviewed additional medical evidence including a copy of Dr. Perper's December 26, 2003 consultation report and the March 10, 2004 consultation report of Dr. Naeye. (EX 2). With respect to Dr. Perper's report, Dr. Caffrey wrote that the report was contradictory with respect to the Miner's smoking history, and Dr. Perper's conclusions were completely erroneous. *Id*. at 2. With respect to Dr. Perper's opinion concerning the Miner's COPD and pulmonary emphysema, Dr. Caffrey "completely disagree[d] with [Dr. Perper's] overwhelming credit of the emphysema that Mr. Wess had to his coal mine experience rather than the miner's cigarette smoking." *Id*. at 3. In contrast to the opinion of Dr. Perper, Dr. Caffrey opined that the Miner's 40-pack years of smoking cigarettes was the major cause of his emphysema and that his coal mine employment "definitely did not play a role in his development of emphysema." *Ibid*. Dr. Caffrey also disputed Dr. Perper's conclusion that the autopsy slides revealed any evidence of complicated coal workers' pneumoconiosis. *Id*. at 3-4. He wrote, in relevant part:

> The autopsy pathologist did not grossly or microscopically describe a lesion of complicated CWP and he did not diagnose complicated CWP. In his gross description he said there were a few scattered coal micronodules less than 0.7 cm and he said there was a single white, non calcified nodule in the left upper lobe measuring 1.2 cm in size; this 1.2 cm size lesion in the left upper lobe happened to be an area of subpleural scarring as I described in my microscopic description on slide labeled "10;" this was not a lesion of complicated pneumoconiosis. . . . If indeed the patient had had a lesion of complicated pneumoconiosis I am sure the autopsy pathologist would have diagnosed complicated pneumoconiosis. I therefore definitely disagree with Dr. Perper's diagnosis of complicated pneumoconiosis. . . .

Id. at 4. Finally, Dr. Caffrey disputed Dr. Perper's conclusion that the Miner's CWP and associated centrilobular emphysema were a contributing cause of the Miner's death both directly and indirectly through pulmonary insufficiency and through hypoxemia which triggered or aggravated an arrhythmia. *Ibid*. The emphysema, he wrote, was caused by smoking, and neither it nor the Miner's mild coal workers' pneumoconiosis caused his cardiac disease with congestive heart failure, his diabetes mellitus, or his atherosclerosis which resulted in the aortic aneurysm which partially ruptured and required surgery from which the Miner never completely recovered. *Id*. at 4-5.

Dr. Richard L. Naeye

According to a March 10, 2004 report prepared by Dr. Naeye, who is board-certified in Anatomic and Clinical Pathology, he reviewed multiple medical treatment records, the

consultation reports of Drs. Caffrey and Perper, the autopsy report of Dr. Turjman, and 15 autopsy slides. (EX 1). He noted upon review of the slides that there was evidence of very old fibrotic macules and micronodules, the largest of which measured 7 mm in one dimension. Additionally, he found lesions, the largest extending for 2.1 cm along a septum into the deeper lung tissues. At its widest point, the lesion was only 4 mm in diameter, and was almost entirely comprised of very old, dense collagen with rare bits of black pigment and a few birefringent crystals of all sizes. None of the lesions showed any sign of recent growth that might indicate they had expanded in recent years. Dr. Naeye found moderately severe to severe centrilobular emphysema and a very acute lobular pneumonia which had reached the early abscess stage at multiple sites. He opined that pneumonia was the direct cause of the Miner's death.

Dr. Naeye diagnosed the Miner with mild, very old coal workers' pneumoconiosis. He made this determination based on the presences of macules and micronodules with very old collagen and no evidence of any inflammatory activity in the Miner's lung tissues. He wrote: "This complete lack of fibroblastic or any other evidence of acute or chronic inflammation at the time of death is important because it excludes the presence of complicated coal workers' pneumoconiosis (CWP) in his lungs." *Id*. at 2. He further noted that complicated CWP was excluded by the lack of evidence of growing CWP lesions on the Miner's chest x-rays over the years which revealed no more than mild CWP. Dr. Naeye concluded that the Miner's CWP was too mild to have caused any disability or to have had any role in his death, and he attributed the Miner's pulmonary disability entirely to his centrilobular emphysema and probable chronic bronchitis. Dr. Naeye described the single 2.1 cm lesion as very old and thin, having long ago grown along an intrapulmonary septum, which had none of the rapid growth features of complicated CWP.

Dr. Naeye gave a deposition on May 4, 2005. (EX 6). He again discussed the 2.1 cm lesion in the Miner's lung tissue. He explained that the lesion was present along a septum in the lung and measured 0.4 cm at its widest point. He opined that the lesion was very old and did not have any characteristic features of growth in recent years. For these reasons, Dr. Naeye determined that the lesion did not have any characteristics of complicated pneumoconiosis. In fact, he concluded that the 2.1 cm lesion was actually a series of small lesions that eventually grew together until they coalesced into a string. Dr. Naeye explained that because the string of lesions was thin, they often failed to appear on the Miner's past chest x-rays.

Dr. Naeye also opined that the Miner's emphysema was caused by his smoking and noted that cigarette smoking has a four or five times greater effect than coal dust in causing centrilobular emphysema. He concluded that the Miner had simple, not complicated, pneumoconiosis. Dr. Naeye further concluded that the pneumoconiosis lesions were too small and too few in number to cause, contribute to, or in any way hasten the Miner's death.

D. Hospital and Treatment Records

Dr. Muljibhai J. Thakkar

Medical records of Dr. Thakkar dated April through June 2000 reflect treatment of the Miner for hypertension, atrial flutter with rapid ventricular response, chronic obstructive

pulmonary disease, and exogenous obesity. (DX 11).

Treatment Records of Clinch Valley Medical Center

Medical records from Clinch Valley Medical Center dated 1996 through 2002 reflect treatment of the Miner by Drs. German Iosif and Muljibhai J. Thakkar for a variety of conditions including , syncope, chronic obstructive pulmonary disease, end-stage bullous emphysema with chronic respiratory failure and cor pulmonale, secondary severe pulmonary hypertension, pneumonia, diabetes mellitus, and hypercalcemia. (DX 12).

Treatment Records of Wellmont Holston Valley Medical Center

Included among the records attached to the October 7, 2003 supplemental report of Dr. Caffrey are records of the Miner's final hospitalization at Wellmont Holston Valley Medical Center dated July and August 2002. (DX 23). A consultation report by Dr. Steven C. Butler dated July 24, 2002 notes an assessment of acute renal failure, status post aortic aneurysm repair, underlying Type II diabetes mellitus, history of hypertension, and atherosclerotic vascular disease. A discharge summary shows that the Miner was admitted on July 22, 2002 with multiple medical problems, and that he had been transferred to that facility for a ruptured abdominal aortic aneurysm where he immediately underwent surgery to repair the aneurysm. The discharge summary further notes that the Miner died on August 6, 2002 after he became bradycardic.

E. Death Certificate

According to his death certificate, James Garnet Wess died on August 6, 2002 at the Holston Valley Medical Center in Kingsport, Sullivan County, Tennessee. (DX 9). The miner was then 82 years of age, married, and survived by his wife. The immediate cause of death was listed as "multiple system organ failure" and the only other condition listed as an underlying cause of death was "ruptured abdominal aneurysm." The physician who signed the death certificate was Dr. Cheryl A. Stanski.

DISCUSSION AND CONCLUSIONS OF LAW

Presence of Pneumoconiosis

As noted above, for a survivor's claim, an initial determination must be made as to the existence of pneumoconiosis. § 718.205(a)(1). The regulations provide four methods for finding the existence of pneumoconiosis: chest x-rays, autopsy or biopsy evidence, the presumptions in §§ 718.304, 718.305 and 728.306, and medical opinions. § 718.202(a)(1)-(4).

In the instant case, Employer does not contest the presence of simple pneumoconiosis. Furthermore, the medical opinions of Drs. Perper, Caffrey and Naeye all establish that the Miner had at least simple coal workers' pneumoconiosis. Also, the autopsy report prepared by Dr. Turjman expressly notes that the Miner suffered from pneumoconiosis. Because of this overwhelming evidence, it is clear that pneumoconiosis existed within the Miner's lungs at the

time of death.

Causation

The second issue in a survivor's claim relates to whether the Miner's pneumoconiosis arose out of coal mine employment. § 718.205(a)(2). Certain presumptions apply to Miners who have worked in coal mine employment for a specified length of time. More specifically:

> If a miner who is suffering or suffered from pneumoconiosis was employed for ten years or more in one or more coal mines, there shall be a rebuttable presumption that the pneumoconiosis arose out of such employment.

§ 718.203(b); § 718.302.

Here, Employer has stipulated that the Miner was employed as a coal miner for at least 23 years. (Tr. 5, 6). Dr. Turjman diagnosed coal workers' pneumoconiosis based on macroscopic and microscopic autopsy evidence which revealed the presence of multiple coal macules and a few scattered coal micro-nodules. Dr. Perper similarly diagnosed coal workers' pneumoconiosis based on micronodules between 0.2 cm and 0.5 cm in size scattered throughout the pulmonary parenchyma which he described as "primarily of the fibro-anthracotic mixed dust type, although a few resemble the silicotic type." Drs. Caffrey and Naeye also diagnosed the miner with simple coal workers' pneumoconiosis. Based on this evidence, I find that the Miner's pneumoconiosis arose from his coal mine employment.

Miner's Death Due to Pneumoconiosis

The central issue in this survivor's claim is, as noted above, whether the Miner's death was "due to" pneumoconiosis. § 718.205(a)(3). For claims filed on or after January 1, 1982, such as this claim, § 718.205(c) provides, in relevant part, that death will be considered to be due to pneumoconiosis if any of the following criteria are met:

> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
> (3) Where the presumption set forth at § 718.304 is applicable.

20 C.F.R. § 718.205(c)(1)-(3). The regulation further states that "[p]neumoconiosis is a 'substantially contributing cause' of a miner's death if it hastens the miner's death." 20 C.F.R. § 718.205(c)(5).

A. *Complicated Pneumoconiosis.*

The Claimant argues that the Miner suffered from complicated pneumoconiosis. Section 718.304, as noted above, provides an irrebuttable presumption of causation of death if a deceased miner is found to have had complicated pneumoconiosis. Thus, under § 718.205(c)(3), a

survivor is eligible for benefits if she can establish that the miner had complicated pneumoconiosis. Complicated pneumoconiosis is established by x-rays that yield one or more large opacities (greater than one centimeter in diameter) and would be classified as Category A, B, or C, or by an autopsy or biopsy, which yields evidence of massive lesions in the lung or nodules in the lung that would equate to a one centimeter or greater opacity on x-ray. The determination of whether the miner has complicated pneumoconiosis is a finding of fact, and the administrative law judge must consider and weigh all relevant evidence. *Melnick v. Consolidation Coal Co.,* 16 B.L.R. 1-31 (1991); *Maypray v. Island Creek Coal Co.*, 7 B.L.R. 1-683 (1985).

An "equivalency determination" is necessary when there is a question about whether nodules found in the lung upon medical examination (autopsy or biopsy) would correspond to opacities viewed on an x-ray indicating complicated pneumoconiosis. In particular, there is dispute over whether a one centimeter nodule or lesion on an autopsy would equate to a one centimeter opacity on a chest x-ray. Some physicians maintain that a chest x-ray will record only the central part of the actual lesion and, therefore, a lesion must be larger than one centimeter on autopsy to constitute a one centimeter opacity on a chest x-ray. Other physicians argue that a one-centimeter lesion on autopsy would equal a one centimeter opacity on a chest x-ray.

In *Braenovich v. Cannelton Industries, Inc.*, 22 B.L.R. 1-236 (2003), the Board upheld the administrative law judge's "equivalency determination" that a 1.5 centimeter lesion on autopsy would constitute a 1.0 centimeter or greater opacity on a chest x-ray, thus establishing the presence of complicated pneumoconiosis under § 718.304. In support of the administrative law judge's finding, the Director argued that the autopsy prosector and a reviewing pathologist found a lesion larger than one centimeter in the miner's lungs. The approach in *Braenovich* is one that has been upheld by the Fourth Circuit. With regards to the use of "equivalency determinations," the Fourth Circuit mandated that administrative law judges "must perform equivalency determinations to make certain that regardless of which diagnostic technique is used, the same underlying condition triggers the irrebuttable presumption." *Double B Min., Inc. v. Blankenship*, 177 F.3d 240, 243 (4th Cir. 1999).

The difficulty with equivalency determinations, of course, is establishing how autopsy or biopsy results can yield evidence of massive lesions in the lung or nodules in the lung that equate to a one centimeter or greater opacity on an x-ray. *Blankenship* noted that for many in the medical community and at the Benefits Review Board, "one lesion of two centimeters or greater in diameter is the *minimum* requirement for establishing 'massive lesions' and thereby invoking the irrebuttable presumption." *Id.* (emphasis added). Nonetheless, the Court refused to impose a two-centimeter pathological rule on the Board, noting that no statute mandates use of the medical definition of complicated pneumoconiosis. *Id.* at 244.

Only Dr. Perper addressed the equivalency principle in his medical opinion report. (DX 26). He wrote, in relevant part:

> The examination of the autopsy lung sections by the current reviewer, revaled [sic] not only macular and micronodular lesions consistent with moderate coal

> workers' pneumoconiosis, but a macronodular irregular lesion of anthraco-hyalino-fibro-silicosis, exceeding 2.0 cm and consistent with progressive massive fibrosis, a size exceeding significantly the more recent requiring [sic] for diagnosing complicated coal workers' pneumoconiosis (progressive massive fibrosis or PMF.). It is true that some pathologists require a 2.0 cm or larger anthraco-fibrotic or anthraco-fibro-hyaline mass in order to diagnose complicated coal workers' pneumoconiosis, according to a recommendation included in a report of the Pneumoconiosis Committee of the College of American Pathologists to the National Institute for Occupational Safety and Health, published in 1979 in the Archives of Pathology and Laboratory Medicine. However, other pathologists, including the current reviewer, consider a lesion of 1.0 cm or larger sufficient to qualify for a nodule of complicated coal workers pneumoconiosis (Progressive Massive Fibrosis). This determination is based on the following facts: - Radiologically observed nodules which are 1.0 cm or larger, are sufficient for a radiological diagnosis of coal workers' pneumoconiosis. There is no reason why a radiological lesion of this size would not correspond to an anatomico-pathological lesion of the same size. The above quoted 1979 report . . . recognized the validity of choice in selecting a nodular size of 1.0 cm as the minimal size for diagnosing complicating [sic] coal workers' pneumoconiosis and stated clearly . . . : "By definition, the lesion (of complicated coal workers' pneumoconiosis) is at least 2 cm in diameter. This arbitrary choice of a minimal diameter does not imply that small lesions do not occur but permits better correlation with clinical and roentgenographic measurements. James chose to use an arbitrary size of 3 cm or greater in defining PMF; others have elected to use 1 cm or more." (citation omitted). In the case of Mr. Wess this is an academic discussion, because the nodule described in the microphotographs with the attached cm. ruler, satisfy even the more stringent 2 cm, requirement for diagnosis [sic] complicated coal workers' pneumoconiosis (PMF.)

Id. at 26-27.

Dr. Naeye agreed with Dr. Perper's assessment that a 2.1 cm lesion existed in the Miner's lung. (EX 1). However, according to Dr. Naeye, the lesion was comprised of very old, dense collagen with rare bits of black pigment and a few birefringent crystals of all sizes, and he noted that the lesion had neither grown nor expanded in recent years and had none of the rapid growth features of complicated coal workers' pneumoconiosis. Because of the lack of any inflammatory activity in the Miner's lung tissue, he opined that the Miner did not suffer from complicated pneumoconiosis. He wrote: "This complete lack of fibroblastic or any other evidence of acute or chronic inflammation at the time of death is important *because it excludes the presence of complicated coal workers' pneumoconiosis (CWP) in his lungs*." *Id*. at 2 (emphasis added). He further concluded that complicated pneumoconiosis was excluded by the lack of evidence of growing coal workers' pneumoconiosis lesions on the Miner's chest x-rays over the years which revealed no more than mild coal workers' pneumoconiosis.

In his deposition, Dr. Naeye further testified regarding the 2.1 cm lesion revealed in the Miner's autopsy slides. (EX 6). He explained that the 2.1 cm lesion did not constitute one lesion,

but instead consisted of a "series of lesions" along the Miner's septum which were only 0.4 cm wide. Dr. Naeye further explained that when smaller lesions coalesce to form the larger lesions of complicated coal workers' pneumoconiosis, they generally coalesce in more or less of a circular or rounded pattern which, when measured, is at least 1 cm in diameter. The lesion in the Miner's case, although measuring 2.1 cm in length, was long and thin, not circular, and measured only 0.4 cm in width. Based on all these facts he concluded that the lesion was not one of complicated pneumoconiosis.

Dr. Caffrey, like Dr. Naeye, concluded there was no evidence of complicated pneumoconiosis and diagnosed, *inter alia*, acute necrotizing bronchopneumonia bilaterally, moderately severe centrilobular and panlobular emphysema, and simple coal workers' pneumoconiosis (noted as "minimal). (EX 2). With respect to the autopsy report of Dr. Turjman, he wrote:

> I definitely disagree with the autopsy pathologist, ... The autopsy slides show minimal or very few lesions of simple CWP, although there are lesions of CWP. My microscopic opinion definitely coincides with what Dr. Turjman's provisional anatomical diagnosis was and that is and I quote: "Possible mild (early state) coal workers' pneumoconiosis."

Id. at 3. (bolding and italics omitted). Dr. Caffrey also disputed Dr. Perper's conclusion that the autopsy slides revealed any evidence of complicated coal workers' pneumoconiosis. *Id*. at 3-4. He wrote, in relevant part:

> The autopsy pathologist did not grossly or microscopically describe a lesion of complicated CWP and he did not diagnose complicated CWP. In his gross description he said there were a few scattered coal micronodules less than 0.7 cm and he said there was a single white, non calcified nodule in the left upper lobe measuring 1.2 cm in size; this 1.2 cm size lesion in the left upper lobe happened to be an area of subpleural scarring as I described in my microscopic description on slide labeled "10;" this was not a lesion of complicated pneumoconiosis. . . . If indeed the patient had had a lesion of complicated pneumoconiosis I am sure the autopsy pathologist would have diagnosed complicated pneumoconiosis. I therefore definitely disagree with Dr. Perper's diagnosis of complicated pneumoconiosis. . . .

Id. at 4.

With respect to the issue of whether the Miner suffered from complicated coal workers' pneumoconiosis, I find the opinions of Drs. Naeye and Caffrey are entitled to greater weight than the contrary opinion of Dr. Perper. Both Dr. Naeye and Dr. Caffrey note that Dr. Turjman, who performed the autopsy of the Miner, did not diagnose complicated coal workers' pneumoconiosis despite having noted the presence of a 1.2 cm lesion in the autopsy slides. Furthermore, Dr. Naeye explained that: the 72 chest x-ray films which he reviewed failed to reveal any evidence of growing coal workers' pneumoconiosis lesions over the approximately 20 years which they spanned; there was no evidence in the autopsy slides of any inflammatory activity in the lungs

consistent with complicated coal workers' pneumoconiosis; and the 2.1 cm "lesion" upon which Dr. Perper relied as evidence of complicated coal workers' pneumoconiosis was actually a long 0.4 cm wide string of lesions which was inconsistent with the rounded or circular lesion which is typical of complicated coal workers' pneumoconiosis. Based on the foregoing, I find the Miner did not have complicated pneumoconiosis. I thus further find that Claimant is not entitled to the irrebuttable presumption under 20 C.F.R. § 718.304 that the Miner's death was due to pneumoconiosis.

B. *Cause of Death.*

With regard to the issue of whether pneumoconiosis was the cause of the Miner's death, was a substantially contributing cause or factor leading to the Miner's death, or hastened the Miner's death, I also find, for the reasons set forth below, the opinions of Drs. Caffrey and Naeye more persuasive than the contrary opinions of Drs. Perper and Turjman.

In his autopsy report, Dr. Turjman wrote: "The presence of extensive bronchopneumonia in both lungs most likely represents the terminal events in this patient with the underlying coal workers' pneumoconiosis being a contributing factor." (DX 10 at 4). The autopsy was performed two days after the Miner died and consisted solely of an examination of the lungs. Dr. Turjman gave absolutely no rationale for his conclusion that the Miner's pneumoconiosis, which he diagnosed as "moderately advanced," was a contributing factor in the Miner's death. Nor did he review the medical records from Wellmont Holston Valley Medical Center where the Miner expired on August 6, 2002. Dr. Cheryl Stanski, who treated the Miner during his final hospitalization, identified the immediate cause of death as "multiple organ system failure" and further noted a "ruptured abdominal aneurysm" as the only condition leading to the immediate cause of death. (DX 9, 23). She noted in her discharge summary that the Miner, who was 82 years of age and had multiple medical problems, had been transferred to Wellmont Holston Valley Medical Center on July 22, 2002 after having sustained a ruptured abdominal aortic aneurysm and immediately underwent surgical repair of the aneurysm. (DX 23). She further noted that he became bradycardic on August 6, 2002 and thereafter expired. *Ibid*. Dr. Turjman's failure to consider this evidence, or to explain the basis for his opinion that coal workers' pneumoconiosis was a "contributing factor" in the Miner's death, renders his opinion of little value. *See Cosaltar v. Mathies Coal Co*., 6 B.L.R. 1-1182 (1984). I find that his opinion regarding the cause of the Miner's death is neither well-reasoned nor well-documented, and thus give it little weight. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(en banc); *Mabe v. Bishop Coal Co*., 9 B.L.R. 167 (1986).

Dr. Perper based his opinion that pneumoconiosis was a substantial contributory cause of the Miner's death in part on his inaccurate characterization of the Miner's pneumoconiosis as "severe" and his faulty conclusion that the Miner suffered from complicated coal workers' pneumoconiosis. Furthermore, although Dr. Perper acknowledged that centrilobular emphysema is a known complication of smoking, he attributed the Miner's respiratory condition to his coal mine employment based on his erroneous conclusion that "the records indicate [the Miner] was only a tobacco chewer" and "it is doubtful whether he was a smoker at any time . . . ." (DX 26 at 27-28). A physician's opinion which is based on an inaccurate smoking history is less probative than other opinions which accurately reflect a claimant's smoking history. *Trumbo v. Reading*

Anthracite Co., 17 B.L.R. 1-85 (1993). Finally, despite the attending physician's identification of the immediate cause of death as "multiple system organ failure" contributed to by a "ruptured abdominal aneurysm," Dr. Perper's only explanation regarding how the Miner's respiratory impairment played any role in causing, contributing to, or hastening the death of the Miner was that his "marked coal workers pneumoconiosis" resulted in "pulmonary insufficiency" and "hypoxemia precipitating/aggravating a cardiac arrhythmia in an individual with heart disease." (DX 26 at 28). This conclusion is neither well-reasoned nor supported by the other medical evidence of record.

In contrast to the flawed opinion of Dr. Perper, Drs. Caffrey and Naeye, as noted above, both gave logical and cogent explanations supporting their opinions that the Miner did not suffer from complicated coal workers' pneumoconiosis. Both physicians also concluded that any respiratory impairment suffered by the Miner was due to his emphysema which was caused by his lengthy smoking history, not his coal mine employment. Dr. Caffrey and Dr. Naeye also described the Miner's simple pneumoconiosis as "mild." For example, Dr. Caffrey concluded, based on the medical evidence he reviewed, that the lesions of coal workers' pneumoconiosis found in the Miner's lungs represented only approximately 2% of the total lung tissue and would cause no respiratory impairment. (DX 23 at 2-3). Similarly, Dr. Naeye concluded that the pneumoconiosis lesions found in the Miner's lungs were too small and too few in number to cause, contribute to, or in any way hasten the Miner's death. (EX 6 at 24). Furthermore, Dr. Caffrey disputed Dr. Perper's conclusion that the Miner's CWP and emphysema were a contributing cause of the Miner's death through pulmonary insufficiency and hypoxemia. He concluded that neither the Miner's emphysema nor his mild coal workers' pneumoconiosis caused his cardiac disease with congestive heart failure or his atherosclerosis which resulted in the aortic aneurysm that partially ruptured and required surgery. (DX 23 at 4-5). Dr. Naeye similarly noted that the Miner had "a very acute lobular pneumonia that had reached the early abscess stage at multiple sites . . . [and t]his pneumonia was the direct cause of his death." (EX 1 at 2). During his deposition, he testified that if the Miner had never worked in the coal mines but had the same smoking history documented in his medical records, he would have died at the same time and in the same manner as occurred on August 4, 2002. (EX 6 at 24). According to Dr. Naeye, the Miner's advanced emphysema, which was caused by his many years of cigarette smoking, substantially destroyed the defense mechanisms in the lungs which would otherwise have controlled the necrotizing pneumonia shown on the autopsy slides which developed following the surgical repair of his abdominal aortic aneurism. *Id*. at 13-15. Both Dr. Caffrey and Dr. Naeye concluded, based on their review of the medical evidence, that coal workers' pneumoconiosis neither caused, contributed to, nor hastened the Miner's death. They, like Dr. Turjman, concluded that the Miner died as a result of pneumonia which developed following surgical repair of his aortic aneurism, although Dr. Turjman opined, without explanation, that pneumoconiosis was a contributing cause of death. I find that the opinions of Drs. Caffrey and Naeye are well reasoned and documented, and entitled to greater weight than the contrary opinions of Drs. Turjman and Perper. I thus find that Claimant has failed to establish that the Miner's death was due to pneumoconiosis.

ENTITLEMENT

Upon consideration of all of the evidence of record, I find that Claimant has failed to meet her burden of proof that the Miner's death was due to pneumoconiosis. She thus cannot recover benefits under 20 C.F.R. § 718.205.

ATTORNEY'S FEES

The award of an attorney's fee under the Act is permitted only in cases in which Claimant is found to be entitled to benefits. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to Claimant for services rendered to her in pursuit of this claim.

ORDER

IT IS HEREBY ORDERED that the claim of Rachel Wess for benefits under the Act is DENIED.

A

STEPHEN L. PURCELL
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefit Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C., 20210.